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Corrosive injuries of the oesophagus and stomach: experience in management at a regional paediatric centre

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In a 21-year period between June 1974 and May 1995, eight children required surgery for the treatment of complications after ingestion of corrosive substances. There were six oesophageal injuries due to alkali ingestion and two gastric injuries secondary to acid ingestion.

Of those ingesting alkali, diagnosis of stricture was made at a mean of 28 days and all children underwent endoscopic dilatation with a mean of six treatments (range 2–13). Two children subsequently required oesophageal replacement and colonic interposition.

One of the children ingesting acid presented as an emergency with peritonitis and required laparotomy at which partial gastrectomy and pyloroplasty were performed. The second child presented 3 weeks after ingestion with a gastric stricture and required gastrogastrostomy.

All children are currently alive and well and are asymptomatic.

The details of management are discussed, together with a review of the literature.

It has been estimated that 40 000 or more paediatric accidental poisonings occur annually in England and Wales, and approximately one-third of these are because of ingestion of household goods *(1)*. Caustic substances account for a small but significant proportion of the more severe cases, with 750 new cases documented per year in Britain, of which about 30 cases are from Wales *(2)*.

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We report the experience of a regional paediatric unit in the management of these injuries over a period of 21 years, during which time eight children have required surgical intervention.

Patients and methods

Between June 1974 and May 1995, eight children were referred to the Department of Paediatric Surgery at the University Hospital of Wales for treatment of complications after the ingestion of corrosive substances. There were five males and three females and the average age was 35 months (range 15–60 months). There were six oesophageal injuries which followed ingestion of alkaline substances and two gastric injuries after ingestion of acidic compounds. The agents responsible are listed in Table I.

Presentation

All children were seen as referrals having first been cared for by a paediatric team. Patients with a history of ingestion of alkali typically presented to the paediatricians

Table I. Agents causing caustic injury

	Products	*Corrosive agent*	*No.*
Alkali (6)	Beer pump cleaning fluid	20% NaOH	4
	Oven cleaner	20% NaOH	1
	Clinitest tablet	30% NaOH	1
Acid (2)	Soldering flux	$ZnCl_2$	1
	Sulphuric acid	H_2SO_4	1

with a history of vomiting (4/6), oral pain (4/6), together with signs of oral ulceration (5/6). In addition, retrosternal pain was reported in three cases as was salivation, and refusal to drink was noted in two patients. However, none of the children reported any abdominal tenderness on examination. They were referred to the paediatric surgery unit with dysphagia which had a mean onset of 28 days after ingestion (range 21–41 days). Children ingesting acid had a different manner of presentation. One child was asymptomatic after ingesting soldering flux and was discharged home after 24 h observation. She then re-presented 3 weeks later with vomiting and weight loss. The second child with acid injury presented with abdominal pain and haematemesis after ingestion of sulphuric acid. On examination he was shocked with a tender rigid abdomen and absent bowel sounds and required emergency laparotomy.

Management

The six patients with a history of alkali ingestion underwent barium swallow investigation which demonstrated evidence of stricture formation in all cases. Strictures were seen at the level of mid-oesophagus in four cases and distal oesophagus in the remaining two cases. Oesophagoscopy with dilatation of the stricture using either bougie or balloon was then performed. Patients in this series required a mean of six dilatations (range 2–13) and two subsequently required surgery. There was a single complication, pneumothorax with surgical emphysema, which occurred after balloon dilatation of a mid-oesophageal stricture. This was treated by insertion of a chest drain and administration of antibiotics and the child suffered no ill-effects. Two cases did not respond to repeated dilatation and required oesophageal replacement. This was carried out by excising the strictured oesophagus and replacing it with a colonic interposition based on the right colic artery. Both children undergoing this procedure required postoperative dilatation, one having six and the other eight treatments, but are currently symptom free.

The child who ingested sulphuric acid required an urgent laparotomy and was found to have a large, ischaemic, necrotic-looking area on the greater curve with a 2 cm central perforation. He underwent partial gastrectomy and pyloroplasty but subsequently developed an antral stricture which necessitated a second operation at which gastroplasty was performed. The second child who had ingested soldering flux re-presented 3 weeks after ingestion with a history of progressive, intermittent effortless vomiting of undigested food together with weight loss. A barium meal was performed which demonstrated a narrowed gastric segment between the antrum and the body in which there was a large posterior ulcer. She underwent laparotomy and a gastrogastrostomy was performed after which she made an uneventful recovery.

We have now developed a protocol for the management of corrosive injuries of the foregut which has been agreed by paediatricians in Wales and which encourages early referral. After a detailed history, including identification of the agent responsible and full examination, the patients undergo chest and abdominal radiographs to exclude perforation of either the oesophagus or stomach. They are then placed on a regimen of intravenous fluids until they are able to tolerate fluids orally and are prescribed broad-spectrum antibiotics for 10 days. An H_2 blocker is also given if damage to distal oesophagus or stomach is suspected. Oesophagoscopy/gastroscopy is also performed if referral has been made within 24 h of ingestion.

Discussion

In excess of 40 000 child accidental poisonings are reported annually in England and Wales, and of these one-third are because of ingestion of household products *(1)*. A small but important number of these are owing to ingestion of caustic substances, and approximately 750 cases are reported annually to the poisons information centres in Britain and of this number around 30 are reported from Wales *(2)*. The male to female ratio of these children is approximately 2:1, and 85% occur in children under 5 years of age. In older children and adults the ingestion of corrosives is usually a suicide attempt as opposed to accidental poisoning *(3)*.

A sharp increase in the incidence of caustic injuries coincided with the introduction of strong alkaline cleaning agents in the 1960s *(4)*. The introduction of legislation to ensure that safety screw tops were fitted to all such bottles has brought a reduction in the incidence; however, many are stored irresponsibly by adults in non-proprietary containers such as lemonade bottles without the safety tops. This was the case in six of eight cases in this series.

The most common initial presenting symptoms and signs in this series were oral ulceration, vomiting and oral pain, while retrosternal pain, salivation and refusal to drink were noted less frequently. None of the children ingesting alkali reported abdominal tenderness. Gaudreault *et al. (5)*, reviewing their experience with a series of 378 paediatric corrosive ingestions, noted that the presence of certain symptoms and signs namely vomiting, dysphagia, excess salivation and abdominal pain were more likely to indicate grade 2 or grade 3 oesophageal burn. However, they did note that interpretation of severity of injury from the presenting symptoms and signs was unreliable. Similarly, Crain *et al. (6)* noted that the presence of two or more of the serious signs/symptoms (vomiting, drooling, stridor and oropharyngeal burns) correlated with endoscopically proven burns, although oropharyngeal burns on their own were an unreliable indicator.

Children ingesting acid had a different manner of presentation. One child was asymptomatic after ingestion of soldering flux and was discharged after 24 h observation. She then re-presented with dysphagia and vomiting owing to gastric stenosis 3 weeks after ingestion. The second child, who had ingested sulphuric acid, presented with abdominal pain and haematemesis and was found to be shocked with a tender rigid abdomen and absent bowel

sounds. A radiograph confirmed a free peritoneal gas viscus and he underwent an emergency laparotomy at which a large gastric perforation was identified.

The role of steroids in the treatment of corrosive injury is controversial. Many of the published series *(4,7–15)* and several experimental models *(16–21)* have advocated their use; however, others dispute their benefits and some question whether their use may mask mediastinitis or peritonitis *(3,22–29)*. There have been only four randomised or controlled trials in humans, of which two identified a benefit from the use of steroids and the other two found no significant difference in the incidence of stricture formation. However, all were small series and the largest was only 60 patients *(3,7,8,26)*. An additional problem in drawing any firm conclusions is that the majority of series contained individuals of all ages and were not limited to the paediatric population. The routine use of steroids is thus not advocated. The use of antibiotics is less controversial because of the risk of mediastinitis or peritonitis which carry a recognised mortality.

We advocate early endoscopy as part of our management protocol if the child is believed to have ingested an alkaline, since it is well-recognised that the presenting symptoms and signs do not always correlate with the degree of oesophageal damage and the absence of oral ulceration does not exclude oesophageal injury *(5,6, 8–11,13–15,23,31–34)*. This policy is recommended by a number of centres *(3–5,7,11,15,22–24,28,30,35–37)*. Some, however, have opposed this because of the risk of perforation *(8,31)*. Endoscopy allows visual assessment of the oesophagus and grading of the extent of injury and it has been shown that the severity of damage correlates with the likelihood of subsequent stricture formation *(7,9,30–32)*. A grade 1 injury has the appearance of mucosal hyperaemia with oedema and superficial erosions. In a second-degree burn there is transmucosal ulceration down to the muscularis mucosae, while in a third-degree burn there is deep ulceration with risk of mediastinal or peritoneal penetration *(31)*. We would not recommend endoscopy if referral is made between 2 and 14 days since the oesophagus is at risk of perforation; however, others believe that the time after ingestion is immaterial and advocate endoscopy in all cases *(3)*.

The process of injury and healing after ingestion of alkali may be divided into three phases *(38)*. The first is damage to tissues by a process of liquefactive necrosis with saponification of all layers of the oesophagus wall. From 5 to 14 days, necrotic tissue resulting from vascular thrombosis begins to slough and the oedema occurring as part of the inflammatory reaction is also at a maximum, thus the oesophagus is at its weakest *(15)*. It is at this time that individuals are most susceptible to infection and thus broad-spectrum antibiotics are administered. The third stage occurs between 2 weeks and 3 months. During this phase the collagen deposited as part of the healing process contracts leading to stricture formation. It is now that individuals begin to report symptoms of dysphagia and are referred for investigation.

The incidence of stricture formation has been documented in several large American series of paediatric corrosive ingestions as being between 0.5% and 5.3% *(4,7,28)*. This series has noted that in Wales, with a population approaching 3 million, an average of 30 cases are reported to the poisons information centre each year, thus over the 20-year period of the review, about 600 corrosive ingestions will have occurred. The catchment area for the Welsh regional paediatric surgical centre is Mid and South Wales and so includes about 2 million of the population. During this time, six oesophageal corrosive injuries have required dilatation at our centre and thus, on a nationwide level, a total of nine strictures may have been expected, giving an incidence of stricture formation of 1.5%. In an attempt to prevent stricture formation, several centres have recommended stenting of the damaged oesophagus with silastic or silicone tubes *(29,39–41)*. However, this approach is not universally accepted and was not used in this series.

Strumboff *(42)*, in a review of 1221 patients, noted the following incidences of complications: mediastinitis (20%), oesophageal perforation (15%), gastric perforation (10%), peritonitis (15%), laryngeal oedema (6%), pneumonia (14%) and fistulation to trachea or aorta (20%). However, this is an adult series in which many were suicide attempts, therefore larger quantities of corrosive were likely to have been ingested. Another recognised complication is that of reflux secondary to fibrous constriction of the oesophagus *(43)*. There is also an appreciable mortality associated with ingestion of large quantities of corrosives such as in suicide attempts, and in a series of 2267 cases over 20 years, Postlethwait *(14)* noted a 13.6% mortality. We have not noted any postoperative complications apart from anastomotic strictures which have responded to repeated simple dilatation.

An important recognised long-term complication is that of development of oesophageal carcinoma in the diseased oesophagus. Several large series have evaluated the incidence of oesophageal carcinoma after corrosive ingestion *(44–48)* and identified 100 cases and an overall average incidence of 2.4%, which is around 1000-fold that of the general population. The average age of presentation ranged between 35 years and 53 years with an average interval between ingestion and tumour presentation ranging from 31–46 years. Of 90 tumours, 68 were in the middle third of the oesophagus and resection was only possible in 30 of the 90 owing to advanced disease at presentation. Therefore, children who have their native oesophagus in place will require life-long surveillance.

The role of endoscopy in the acute phase of acid ingestion is less well-defined since the number of cases is far less than for alkaline injuries. Acidic burns result in a coagulative necrosis and eschar formation which limits the depth of penetration. The acid tends to pass quickly down the oesophagus to the stomach then follows the lesser curve to the pylorus where it pools leading to pylorospasm. Therefore, this is the most common site of ulceration, stricture and perforation *(9,35,49–53)*, though these complications may also occur in the oesophagus *(6,54)*. The long-term complications of acid

ingestion are also less well-documented and it is not known whether there is a risk of development of gastric carcinoma.

The treatment of oesophageal stricture is initially by dilatation. This may be performed using Eder–Puestow, Celestin or balloon dilators under fluoroscopic screening. Dilatation was used in all six cases of corrosive stricture in our series and in each case multiple dilatations were required with a range from 2 to 13 procedures. There was a single complication, namely of pneumothorax and surgical emphysema. This occurred on attempting to pass the paediatric fibreoptic endoscope through a stricture after what appeared to have been a successful dilatation. This is a well-recognised complication and was noted by Appleberg *(55)* in 14 of 182 patients undergoing dilatation of strictures.

In the case of oesophageal injury, if the damage is extensive at initial endoscopy or there is evidence of complication such as fistula formation or mediastinitis then an emergency thoracotomy should be performed *(40,56,57)*. In these cases a cervical oesophagostomy may be performed or the diseased oesophagus may be resected with immediate reconstruction. Oesophageal replacement is also indicated in the presence of strictures that are not responsive to repeated dilatation therapy. In these cases options for oesophageal replacement include interposition of stomach *(58,59)*, jejunem *(60,61)* or colon *(62–66)* and the organ may be placed subcutaneously, retrosternally or replaced in the posterior mediastinum. It has been shown that keeping to the posterior mediastinum is the shortest option and this is of particular importance in children *(67)*. We prefer to utilise a colonic interposition based on the right colic artery with the graft being placed in the posterior mediastinum.

The indications for surgery after gastric corrosive injury are similar to those of oesophageal injury, namely emergency laparotomy in the case of uncontrolled bleeding or peritonitis and routine operation for the long-term complications such as stricture. The literature on gastric injury is less extensive than for oesophageal corrosive damage, since acid ingestion, which primarily affects the stomach, is far less common than alkali ingestion. In the acute situation there may be massive haemorrhage or perforation necessitating local resection of necrosed tissue such as our first case. With regard to the development of strictures, these should be investigated in the same way as oesophageal lesions, namely with barium studies and endoscopy *(52)*. If a stricture is identified, then it should be excised in the simplest manner in order to relieve obstruction *(68)*.

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